Tracheoesophageal fistula and tracheomalacia due to elevated endotracheal tube cuff pressure. A case report

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Abstract

Introduction: Pressure monitoring of the endotracheal tube cuff or cannula in prolonged mechanically ventilated patient is essential, in fact, the over-inflation of the cuff may result in deleterious consequences.

In this case report, the patient developed tracheoesophageal fistula on lengthened mechanical ventilation due to excessive pressure of the cuff leading to tracheomalacia. The diagnosis was made only after the onset of complications, pneumonia, abdominal distension and confirmed by bronchoscopy and CT scan of the neck. Critical state of the patient did not permit surgery. The prevention seems to be fundamental, through a rigorous control of the cuff pressure. The literature seems to agree that the pressure limit should not exceeded by 25 cmH2O, in mechanically ventilated critically ill patients for a long time. This limit represents the correct balance between avoiding air leaks around the cuff or the descent of secretions into the trachea, and to avoid the emergence of complications such as fistula and tracheomalacia.

Keywords: Tracheoesophageal fistula,Tracheomalacia, Mechanical ventilation, Cuff pressure.

Introduction

The cuff of the endotracheal tube provides a closed system to permit effective ventilation and airway protection. Cuff pressure should be between 20 - 25 cm H_2O, under inflation is reported to promote leakage of secretions around the cuff, that can develop pneumonia and over inflation of the cuff in prolonged endotracheal intubation, can cause tracheomalacia, tracheal stenosis and tracheoesophageal fistula [1].

Acquired or secondary tracheomalacia in adults is a condition in which there is a weakness of the tracheal wall caused by degeneration of cartilaginous support and hypotonia of the myoelastic elements. It is caused by pressure necrosis, with impairment of the blood supply, infections, and mucosal damage caused due to friction [2].

A relatively unusual complication, occurring in less than 1% of patients undergoing tracheostomy, is the tracheoesophageal fistula (TEF), that is a connection between trachea and esophagus. The excessive cuff pressure of the cannula or tube can cause posterior tracheal wall injury and may also be associated with tracheomalacia.

The acquired type of tracheoesophageal fistula might be caused by malignancy or non malignant entities. The non malignant form accounts for more than 50% of acquired TEF and is a devastating complication [3].

Etiologic factors accounting for acquired non malignant TEF include blunt penetrating trauma, infections, mediastinal infections, esophageal surgery, tracheal surgery, stents, but the most common cause is prolonged mechanical ventilation [4].

We present the clinical case of a patient who developed tracheoesophageal fistula, and tracheomalacia due to excessive cuff pressure during prolonged mechanical ventilation.

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Case report

A 67 years old man was admitted to our intensive care unit for a cerebral hemorrhage. The patient was immediately sedated and intubated with cuffed tube inflated and underwent artificial ventilation for severe impairment of neurological functions, coma and quadriplegia. After eight days of no significant improvement of the clinical picture, a dilatative percutaneous tracheostomy with horn is packaged under bronchoscopic vision, with cuffed tracheostomy tube placement (high pressure, low volume). Mechanical ventilation is continued for 20 days with cuffed tracheostomy tube, and without monitoring of cuff pressure resulting in abdominal distention, typical signs of pneumonia aspiration, alterations in ventilatory parameters measured with a discrepancy between inspiratory and expiratory volume.

The CT scan (Fig. 1A, 1B) and a bronchoscopic examination (Fig. 2A, 2B) shows the presence of a voluminous tracheoesophageal fistula in the insertion area of the cuff of the tracheostomy tube, together with a remarkable tracheomalacia. Dynamic bronchoscopy highlights a significant bulging of the pars membranacea of the trachea, with the characteristic feature saber for the remarkable

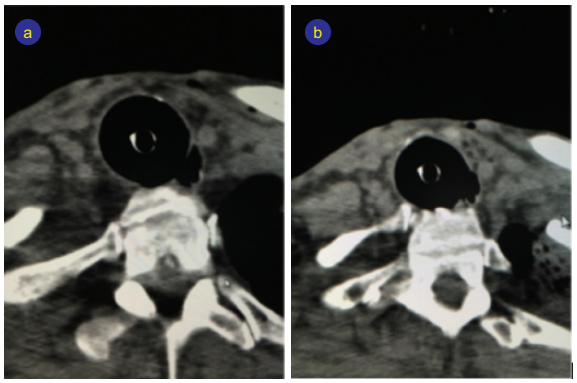

Figure 1a & 1b: image of the fistula with neck CT scan

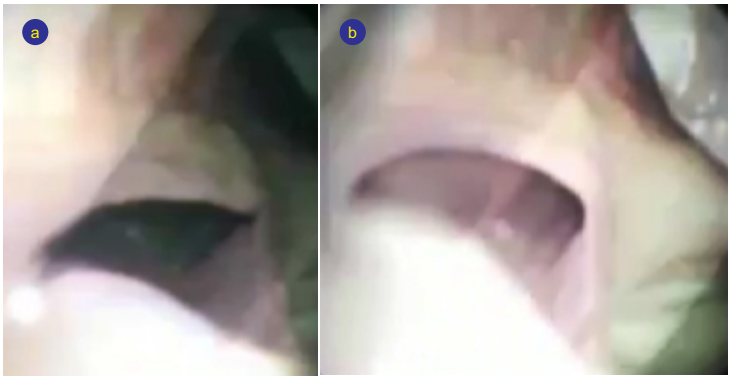

Figure 2a &2b: image of the fistula with bronchoscopy, nasogastric tube.

malacia.

The high cuff pressure on tracheal wall associated with the tracheostomy led to the increase in tracheomalacia, which was preexisting due to long standing COPD. Moreover, the high cuff pressure on tracheal posterior wall caused hypoperfusion and ischemia of the tracheal mucosa, associated due to the direct damage and allowed the development of the tracheoesophageal fistula.

For patient's morbid clinical condition has not been enabled any surgical indication.

Discussion

In 1963, Baxter and Dunbar described tracheomalacia as a weakness of the large airway's wall due to softening of the cartilage with hypotonia of the myoelastic elements [2].

Mair and Parsons [5] described three distinct types of major airway collapse (MAC), type 1 is congenitally present in infants or children and does not involve external airway compression. Type 1 is seen in some premature infants and in children with associated congenital anomalies such as esophageal atresia, tracheoesophageal fistula, mucopolysaccharidosis, and Larsen's syndrome. Major airway collapse type 2 is seen in infants or children with abnormalities involving extrinsic tracheal and or bronchial compression and, consequently, may be congenital or acquired. These include cardiovascular anomalies (eg, double aortic arch, late takeoff of the innominate artery and left atrial hypertrophy), bronchogenic cysts, teratomas, cystic hygromas, hemangiomas, pectus excavatum, severe scoliosis, thymus enlargement and goiter. Major airway collapse type 3 is an acquired malacia arising from prolonged increased ventilatory airway pressures, from tracheotomy or inflammatory processes causing severe tracheobronchitis.

Tracheomalacia frequently is associated with tracheoesophageal fistula. Pathogenic mechanism is represented by the chronic trauma of prolonged tracheal intubation. The pressure resulting from the hyper inflated endotracheal tube cuff on the posterior membranous wall, most often against a rigid nasogastric tube produces ischemic necrosis that also affects the anterior wall of the esophagus, with the result being an abnormal communication [1, 6].

In 1967, Fledge [7] described the first report of TEF caused by cuff related injury in patients who had been ventilated mechanically.

In 1968, Grillo et al. [8] collected tracheal samples of thirty patients during autopsy for analysis. All patients were ventilated with inflated cuff at the time of death, when possible, the tracheas were taken with cuffed tubes inserted to assess the injuries. The length of mechanical ventilation varied from 1 to 8 weeks. tracheitis with fibrin deposition were the first lesions found in the area of the cuff followed by small ulcerations that increased with the length of the ventilation until exposing the tracheal rings. In the final stages, there is a laxity and fragmentation of the rings with bulging of the trachea due to the pressure of the cuff. Also, the trachea at cuff site was completely free of cartilage in the final stages. The exposure of tracheal rings was observed in a time period from 10 days to 4 weeks. Prolonged respiratory assistance by positive pressure ventilation via cuffed tracheostomy or endotracheal tube can be complicated by mucosal erosions, tracheal stenosis, tracheomalacia and tracheoesophageal fistula. Based on histopathological studies, the excessive inflation of the cuff causes ischemia of the tracheal mucosa with the development of tracheal mucosal damage, that can be seen as a direct consequence of tracheal mucosal hypoperfusion, caused by an excessive pressure. Blood flow in the antero-lateral part of the trachea has been reported to be compromised at pressures exceeding 30 cm H_2O and obstructed at pressures exceeding 50 cm H_2O in normotensive patients. The membranous posterior tracheal wall, however, shows less evidence of reduction in blood flow because it is more distensible than the cartilaginous anterolateral [10].

Current evidence suggests that the minimum volume of air to obviate air-flow past the cuff, up to a maximum pressure of less than 25 cm H_2O, is probably the safest practice to minimize high tube cuff pressures.

A wide range of pressures between 20 and 40 has been reported to be safe [11]. Some authors recommend a pressure of 25 cmH2O as a secure pressure to prevent aspiration, air leaks around the cuff and tracheal injury [12-14].

On physical examination, there was abdominal distension caused by inflation of the gastrointestinal tract through the fistula under positive pressure ventilation. Recurrent pneumonia can develop, and there might be a difference between the inspiratory and expiratory tidal volume. Patients extubated might have increased secretions, coughing while swallowing and there might be liquid or food in the expectorated material.

CT and flexible bronchoscopy are the main diagnostic exams for the localization of the fistula, while the dynamic bronchoscopy is used to assess the degree of tracheomalacia.

The tracheoesophageal fistula treatment is very complicated, surgical management is possible only when the patient is not ventilated mechanically and his medical condition has improved. In mechanically ventilated patients, surgery is contraindicated due to the negative effects of the tracheal tube on anastomosis and esophageal suture. In these patients, conservative treatment is suggested subsequent to removal of nasogastric tube. Oral feeding should be avoided and enteral feeding reinstated through gastrostomy or jejunostomy. The tube cuff must be placed downstream of the fistula endoscopically for placement of stents, where surgery is contraindicated.

In adults, tracheomalacia rarely requires therapeutic intervention, if the patient is spontaneously breathing it can recourse to a non-invasive ventilation only if the episodes of bronchostenosis are very frequent, while in the intubated patient tracheomalacia requires no specific treatment, because the cannula or tube makes for gaping the tracheal lumen [9].

In our case, prevention was essential as the patient has not had surgery for serious conditions.

Conclusions

Our case report is an example of the damage due to excessive cuff pressure exerted on the tracheal wall. Hence, a careful monitoring of inflation pressure of the cuff is mandatory to prevent ischemic lesions of the trachea.

For reducing the risk of damage to the tracheal wall during prolonged mechanical ventilation, it is recommended to use tracheostomy tube cuff with low pressure and high volume, also regular cuff pressure monitoring is important so that the pressure does not exceed 25 cm H_2O.

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